finger tip to chest, if the infection is in the finger, and absolute rest in bed on light diet. Heat applied off and on accomplishes comparatively little. Constant heat for twenty-four to forty-eight hours practically clears up the local process. What remains in the lymph glands or more distally will then yield to dry heat. Wet heat continued longer than forty-eight hours, reduces the resistance of the tissues. The wet heat should be given in such a manner that the limb can be elevated. This is best accomplished by wrapping the parts in hot towels. Some antiseptic solution such as lysol prevents the development of pimples. Six to eight bath towels wrapped about by several layers of wool will keep the limb hot for eight to ten hours at a stretch.

Treatment of a general infection is quite a different procedure. Unfortunately it is not so clear cut. Here the local wound is of practically no concern and the whole effort must be directed towards aiding the general bodily reserve. Recently the reports of use of various chemicals introduced into the circulation have raised our hopes, but the practical experience of the average man has been universally disappointing. Many cases have been saved and in most of these instances the effects of administration of the chemical have been nothing less than miraculous. Other patients have gone on and died and sometimes death seems to have been hastened by the reaction to the administration. There is much to be learned. Perhaps the dosage is insufficient, perhaps the poisonous effects of the organisms have left the human organisms too weak to recover even after the organisms have all been destroyed.

Dr. Barnes is to be complimented on his paper. He has taken up the study of the organisms and their mode of action, together with the study of the human physiology, during the operation of general infection. He is on the right track because thorough understanding of these principles will solve the problem of general infection and make our treatment as clear cut as the local process.

In the meantime the immediate task before us is the prevention of a general infection. There is very rarely any excuse for local infection becoming general while under the care of a physician. Proper treatment of the local condition will do away with practically all general infections excepting those which are well established when first seen by the physician.

LUPUS ERYTHEMATOSUS ACUTUS DISSEMINATUS

REPORT OF A FATAL CASE

By FRED B. CLARKE,* M. D., *Long Beach;*
A. W. WARNOCK,* M. D., *San Pedro*

DISCUSSION *by V. R. Mason, Los Angeles; Samuel Ayres, Jr., Los Angeles.*

LUPUS ERYTHEMATOSUS ACUTUS DISSEMINATUS is, if one may judge from the literature, an exceedingly unusual condition. Scholtz, having reviewed the literature in 1922, found reports of twenty-seven cases, and there have been several cases reported since then by Goeckerman, Robertson and Klauder, and Pernet.

It is probable that this condition is of much more frequent occurrence than the number of reported cases suggests, because of the fact that the skin manifestations resemble, in some cases, such diseases as scarlet fever, erysipelas, pellagra, exfoliating dermatitis, spotted fever, etc., and the constitutional symptoms may stimulate such acute infections as rheumatism, malaria, typhoid, etc. The diagnosis of rheumatism was made during one of the five attacks in this case, while during two subsequent attacks, the diagnosis was typhoid.

Review of various case histories suggests that the chronic disseminate type, occurring in an individual who has had the chronic discoid lesions for a long time, is the one most frequently encountered, and that the acute type, beginning without evidence of a previous chronic type of lesion, is exceedingly rare, and usually runs a course suggesting an acute septic condition.

Our case began without the characteristic chronic lesions, but with a history of four previous illnesses, during which there was a persistent fever, with mild arthritic disturbances and a skin lesion diagnosed as erythema nodosum, suggesting the constitutional background upon which the acute type of cases develop was present.

The underlying condition producing hypersensitiveness in these patients is a thing in which we are primarily interested, because of their reaction to toxins and to the removal of foci of infection, hypersensitiveness to tuberculin, etc. In addition the high mortality rate, once the condition is developed, is important. Text books devote but little space to the acute type, and the following classification by Robertson and Klauder would seem a good one:

"All varieties of lupus erythematosus appear to fall into four clinical groups:

"Circumscribed or discoid form, chronic, occurring chiefly on the head and face, especially on the nose, cheeks, and lobes of the ears. Diffuse or disseminated, of which there are three varieties: (a) Disseminated, but not acute; (b) Disseminated, acute, developing from chronic discoid form; (c) Acute from the beginning, rapidly becoming disseminated and running an acute course throughout.

"The disseminated but not acute, may be associated with mild constitutional symptoms which some writers style subacute disseminated lupus erythematosus, and others erroneously designate as lupus erythematosus acutus disseminatus. This group is more commonly seen than the pure type of lupus erythematosus acutus disseminatus. Patients in this group are doubtless potential candidates for the acute disseminated form of lupus. It is stated that the presence of albuminuria is of bad prognostic significance.

"Group (b), disseminated, acute, developing from the chronic discoid form of lupus and group (c), acute from the beginning, rapidly becoming disseminated, may end fatally in a few weeks or months. These cases, and particularly those falling in group (c), are further characterized by increasing evidences of constitutional involvement, partaking of the nature of some acute general infection. The two latter forms of lupus present a fairly uniform eruption on the face and palms, and a multiform eruption on the trunk and extremities. These are the pure types of lupus erythematosus acutus disseminatus, the lupus erythematosus aggregatus universalis (erysipelas perstans facei of Kaposi), and group (c), the acute

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lupus erythematosus aigu d'emblé of some writers, notably Pernet."

CASE HISTORY

J. L. H., male, age 34, white, married, a lumber surveyor, when first seen on the third day of his illness, complained of a stiffness of joints, nausea, general malaise, and anorexia, with the history that two weeks before the onset of the present illness, an impacted molar tooth had been extracted.

The skin showed no abnormalities except a slight generalized cyanosis and a coldness to the touch. His hands were stiff, especially the left, which he could not close, due to a previous synovitis. The spine and lower extremities were normal. The glands of the neck, axilla, and groin were palpable but not immoderately enlarged nor tender. A slough was present in the cavity of the lower, left, first molar, which had been removed two weeks before. The tongue was coated, the breath foul, but no lesions of the mucous membrane were found. The throat, lungs and heart were negative. The abdomen was negative with the exception that the spleen was enlarged and slightly tender, and the notch was felt about 6 cms. below the costal margin. The temperature was 102.6° by the rectum. The pulse was 80, full and regular, and the respirations 16, and normal in character. Blood: 80 per cent hemoglobin (Dare), 2300 white blood cells, 51 per cent polymorphonuclear leucocytes, 49 per cent lymphacytes (equal numbers of large and small), and no abnormal red or white cells. Two blood cultures were negative; Widal negative; urine negative except slight evidence of bile and albumin; Wassermann negative.

Within eighteen hours, a few maculo-papular lesions, which did not fade on pressure, had appeared under the right breast, but were not present elsewhere on the body. Within three hours these lesions spread all over the chest, abdomen and back, with some on the cheeks and lower forehead, but was not present in the axillary lines, nor lower extremities. The skin was taking on a slightly icteric tinge. By this time the throat became sore, and whitish lesions, not unlike masses of Koplik's spots appeared at the gingivo-labial angle. Smears from these showed, among numerous other organisms, Fuso-Spirallary organisms. The mouth became so painful that the patient was unable to swallow. By the fifth and sixth days the temperature dropped to normal, but the restlessness and insomnia continued. The prepuce and eyes showed a purulent discharge containing staphylococci and a few Gram-positive diplobacilli, and yellowish crusts appeared about the nose and the rash spread to the upper parts of the thighs. Blood count at this time showed 4,530,000 red blood cells, 3200 white blood cells, 81 per cent polymorphonuclear leucocytes, no pathologic cells.

The onset of constitutional symptoms of an indefinite nature accompanied by an unusual eruption, with rather characteristic involvement of the nose and face, combined with the history of previous attacks of an undetermined nature, accompanied in two instances at least by some sort of skin lesion, particularly on the hands, followed by a long convalescence, was, we thought, quite sufficient to justify a diagnosis of lupus erythematosus acutus disseminatus. Within thirty hours the left cornea perforated. The jaundice became very marked, and the patient was having frequent, copious, frothy, clay-colored stools. He was delirious at times, having delusions and hallucinations. The next two days the daily temperature fluctuated between 100 and 103.5°. The rash became practically confluent on the lower extremities, and the lesions of the trunk were covered with fine white scales, but the true character of those of the face was obscured by the abundant beard and the vaseline used to soften the crusts and prevent adhesion of the eyelids. There were well marked hemorrhagic crusts across the bridge of the nose and the malar portions of the cheeks. The lesions covered the entire body, except the scalp, the palms, and soles, though present in decreased numbers in the axillary lines. Ultraviolet radiation was given to which the patient reacted violently. On the fourteenth day he was given 0.3 grams of neoarsphenamin intravenously. Since a slight improvement was noted, this was followed by 0.45 grams the next day.

Bronchial pneumonia developed and death occurred twenty days after the onset.

Autopsy twelve hours later: After the beard and crusts had been removed from the face, the character of the facial lesions was more definite. On each cheek was a spot about 3x4 cm., extending over the bridge of the nose in typical butterfly fashion. Removal of these crusts revealed raw surfaces where the skin had been destroyed.

The viscera were all intensely bile-stained, and there was marked enlargement of the liver and spleen. Numerous small, hard, darkly pigmented glands one-eighth to one-quarter-inch in diameter were found in the omentum and the mesentery of the large intestine. The glands of the small intestine did not show this pigmentation.

Doctor Jean Oliver of the Department of Pathology, Stanford University Medical School, made a complete study of the specimens, but found no evidence of tuberculosis, and reported the changes in the skin as follows:

"Sections of the skin show a marked atrophy of the epidermis with thinning of the epithelial layer and obliteration of the papillae. In some places there is a hypertrophy of the horny layer; in others it is of normal thickness. Occasionally the epithelium is entirely missing, leaving a bare surface. In the dermis and corium there are evidences of a chronic inflammatory process consisting of an increase in the connective tissue, but very little 'round cell' infiltration. In one section there is a small abscess near a hair follicle, otherwise nothing significant."

MORPHOLOGY

Lupus erythematosus acutus disseminatus resembles the erythema multiforme type of dermatosis, and there is a decided tendency to consider this condition as belonging to that group. Certain it is that the type of eruption may and usually does vary decidedly in different cases, and may present almost any type of eruption from a macule to bullae, or there may be purpuric oozings or telangiectatic lesions such as in the case reported by Robertson and Klauder. In this case the lesions began on the breast and were of a bright red maculopapular type, which spread in great numbers within three hours over the chest, abdomen, back and neck, with but few on the face. They were maculopapular in character, bright red, distinctly elevated above the surface, and firm to the touch, remaining for the most part discrete, not fading upon pressure, and persisting with fresh outcroppings on the lower abdomen and legs. The case resembled spotted fever more than any other disease. The eruption over the trunk and extremities changed but little, except to become a darker red. In the older lesions, a fine, yellowish white scale was present, although over the legs a new crop developed without the earlier ones fading away. Upon the face the lesions were different, those over the nose, cheeks and forehead beginning as a macular eruption, which soon coalesced, becoming raised and covered with dark red, scaly crusts, undoubtedly due to capillary oozing. These incrustations were more marked on the nose, and extended over both cheeks, and to a lesser extent over the forehead. The blood-stained incrustations over the nose and cheeks formed a solid mass with no normal areas of skin. Loose, scale-like crusts continued to be formed, and at necropsy, when the entire crust was removed, almost complete destruction of the skin was found, although there were a few irregular islands of the inner layer of the skin remaining. The lips were covered with bloody crusts which the patient rubbed away occasionally, leaving an oozing surface.

Over the right breast and on the right arm, there were two areas 3.5x4.0 cms., over which there was a large, thick, brownish lesion, raised 5.3 cm. above the surface.

Over the forehead were rather dirty, grayish crusts, which tended to disquamate. The scalp was entirely free from lesions. Scar tissue and areas of alopecia were not found, indicating previous lesions.

The palms of the hands and soles of the feet were free from lesions.

SYMPTOMATOLOGY

From case histories, the acuteness of the onset, severity and progression of skin lesions and constitutional symptoms vary widely. Temperature is present in the acute cases, and in this instance was not unlike typhoid in its step-like rise; in fact a diagnosis of typhoid had been made during two previous attacks, and the patient stated that "you need not call this typhoid, as I have been placed in the hospital and had blood cultures and Widals made several times, before the doctors could be convinced that I did not have typhoid." The temperature after the first week became irregular, and was what one might expect in a septic condition.

The leucopenia was present on the first day of temperature, being 2300, but became gradually higher, as the septic condition increased, reaching 7000 before death, with a normal differential count. Leucopenia seems to be a rather constant finding, and in a case reported by Goeckerman was 1800.

Jaundice occurred early and became gradually deeper, until the patient became intensely icteric. We have not noted this in other case reports.

Delirium occurred on the tenth day, similar to the low muttering type of typhoid, becoming gradually more marked until death.

Ulceration of the cornea occurred on the ninth day and, as far as we could determine, it did not result from trauma, but seemed to be the result of an infection of the cornea which finally perforated, producing an extrusion of the contents of the anterior chamber. Smears showed some pus cells, a few staphylococci, and an occasional Grampositive diplobacillus.

Early there was marked purulent discharge from under the prepuce, which was abundant and contained staphylococci and diplobacilli.

Stools on the eighth day became gray, and six days before he died they became voluminous and butter-like in character, denoting marked pancreatic insufficiency.

Because of the severity of the lesions in the mouth, food could not be given although local applications of butyn, anesthesin, etc., were used. Rectal feeding was resorted to until the patient became mentally confused, after which he took liquid nourishment fairly well, until three days before death.

PATHOGENESIS

The pathogenesis has never been satisfactorily determined. Glandular tuberculosis has been, for a long time, considered an essential etiological factor, but many cases do not show evidence of tuberculosis at necropsy, although there undoubtedly does exist in the majority of cases autopsied sufficient evidence of tuberculosis to warrant this factor being of decided importance. Clinically, active tuberculosis is not a necessary feature upon which a diagnosis should be based, as it is not demonstrated in the average case. It is interesting to observe the warnings against the use of tuberculin as a diagnostic or theraputic agent because of the marked susceptibility which some of these cases exhibit. Ravogli has reported a fatal case from the administration of .001 mg. of tuberculin as a diagnostic measure.

Stokes states that "glandular tuberculosis has been prominent in cases studied by him, and suggests the hypothesis that, when septic infection creates the hypersensitiveness, the result of a hematogenous infection by tubercle bacilli is a tuberculid, while when tuberculosis creates the hypersensitiveness the result of a streptococcal or septic invasion is disseminate lupus erythematosus."

The clinical picture presented in this case was that of a steadily progressing sepsis, against which the patient did not seem to have any resistance. The occurrence of nephritis as pointed out by Keith and Rowntree is of interest as being either an accompaniment or an etiological factor in this condition. Scholtz considers that pyonephrosis was the casual factor in his case. Our case has a persistent trace of albumin with an occasional hyalin and granular cast, and we have concluded that the kidney did not present any abnormality not easily accounted for by the general toxemia.

The outstanding thing which seemed to initiate the symptoms in this case was the extraction of the left lower first molar. No evidence of healing was present at the onset of temperature two weeks later, although a curetment of the alveolar fossa had been done one week after extraction.

Smears invariably showed the ordinary bacteria found in the mouth with a few fuso-spirallary organisms, but at no time was there a predominating organism.

Goeckerman cites three cases in which removal of a tooth had resulted in a stuporous state.

Treatment is usually not of particular value in altering the course of the acute type. In this case quinine and sedatives were used early. Neoarsphenamine was used in an effort to clear up the condition of the mouth.

Goeckerman, assuming a glandular tuberculosis, treated one of his cases with deep roentgen ray therapy, with excellent results. In this patient, because of the severity of his symptoms, x-ray was not used.

DISCUSSION

V. R. Mason, M. D. (Pacific Mutual Building, Los Angeles)—The authors have summarized the literature and stated many of the interesting problems presented by this disease. I have been struck by the similarity of the clinical picture presented by the reported instances of the disease. The only important variable seems to be the duration of the malady. This circumstance in the present state of our knowledge favors the assumption that we are dealing with a clinical entity and not with a peculiar reaction to a variety of causes. The patients seem to have acquired increased susceptibility to tuberculin, vaccines and minor infections and I believe we should consider this phenomenon as an important symptom of the disease. Moreover, if this is true, it would explain the frequency and seriousness of secondary com-

plications which are usually the direct cause of death of patients suffering from the malady.

It has been observed and confirmed that in certain instances of this disease human tubercle bacilli may be present in lymph glands which are not the seat of tubercle formation. This may be explained by the unique "hyper-allergic" reaction of these patients to tuberculin. In such instances we should expect marked constitutional reaction with little tubercle formation.

SAMUEL AYERS, JR., M. D. (Westlake Professional Building, Los Angeles)—The acute disseminated type of lupus erythematosus is a rare disease and my personal experience with it is meager. One of the most striking facts is the apparent inadequacy of pathological findings. Two recent cases with necropsy findings reported by Keefer and Felty showed abdominal tuberculous glands as the only pathological findings of note, and in one of these cases tubercle bacilli of the human type were recovered from a lymph gland, which did not show the histologic lesions characteristic of tuberculosis.

Foci of infection of a non-tuberculous type certainly play an important etiological role in the ordinary subacute or chronic types of lupus erythematosus. I have one striking example of this in a woman who developed a rather acute facial lupus erythematosus with classical symptoms, in whom chronically infected tonsils were found After persisting some months the eruption cleared immediately following tonsillectomy—in fact before the patient had fully recovered from the operation—and has not recurred during the past three years. All possible foci of infection should be searched for in all types of lupus erythematosus—tonsils, teeth, sinuses, prostate, intestinal tract, kidneys, bladder, etc., as well as tuberculous glands and other tuberculous foci.

CLINICAL ASPECTS OF INTESTINAL PROTOZOIASIS

By ANDREW BONTHIUS,* M. D., *Pasadena*

Pathogenic parasitic protozoan infections, like bacterial infections, often exist indefinitely in a host without producing symptoms; often they cause such mild symptoms as to escape complaint by the patient; and often the chronic or secondary symptoms are so atypical and indefinite, and so far remote from the actual seat of infection, that the etiologic cause may escape the attention of the physician.

The presence of pathogenic protozoa or their cysts is the only sure proof of the infection.

No patient can be declared cured of intestinal protozoiasis unless, in addition to clinical improvement, the stools remain negative upon successive periodic examinations for at least a year.

DISCUSSION *by H. E. Butka, Los Angeles; R. Manning Clarke, Los Angeles; Herbert Gunn, San Francisco, and John V. Barrow, Los Angeles.*

IN this paper I purpose to discuss chiefly the secondary manifestations, diagnosis, and treatment of intestinal protozoiasis. The parasitic protozoa included in this consideration are the amœba dysenteriæ, balantidium coli, giardia or lamblia, chilomastix, trichomonas, pentatrichomonas, cragia, and councilmania. Each of these, independently or together with one or more of the others, can and often does cause strikingly similar secondary manifestations.

"Entamœbic dysentery is an acute or chronic specific disease of the intestine, caused most commonly by entamœba histolytica, but in some instances, possibly, by other species. These entamœba enter the intestines with food or water and produce colitis and extreme enteritis, characterized by the passage of frequent motions, which generally contain blood and mucus and are associated with abdominal pain and tenesmus. At times they also produce abscesses in the liver and other parts of the body." One may quite correctly add that the above-mentioned parasites, alone or together, may cause the same clinical symptoms.

This definition by Castellani is quite similar to definitions given by several other authors. It is satisfactory for the acute type, but very often the chronic type, or secondary stage, is not characterized by all or even by any of these symptoms. Not every patient whose intestinal tract is invaded by these parasites develops dysentery, severe, mild, or at all. Many go through what may be termed the first stage of invasion by amœba without any, or at most, insignificantly mild, symptoms, and pass on to what I choose to call the secondary stage with its manifestations.

All authorities agree that, under certain conditions, focal infection may exist from one to twenty or more years, during which it may be latent, intermittently active, and later continuously so, depending upon the changed virulence of the invader, and the loss of immunity by the patient, or both. Secondary lesions of focal infection may occur so slowly as to induce no symptoms, and the patient may be unaware of anemia, nephritis, endarteritis, until an advanced stage of invalidism is reached. Rosenow has proved that streptococci in focal infections are not only pathogenic but often possess truly remarkable specificity, for certain organs or cells of the body, as the gall-bladder, stomach, appendix, muscles, kidneys, nerves or heart.

It has been generally accepted as true that bacterial infection may exist indefinitely in a host before producing clinical symptoms and causing symptoms far remote from the actual seat of infection. Why can this not logically hold true for pathogenic parasitic infections also?

From my observations of a series of seventy-four cases with positive parasitic findings, I am of the opinion that bacteria and parasites invade and effect their hosts much alike. This is particularly true of the type of infection most frequently encountered in Caucasians in temperature climates.

My seventy-four patients included those from 3 to 70 years of age; thirty-three were females, forty-one males; forty-nine had lived from one to thirty years in the Orient, two had spent considerable time in Europe, the others had never been out of the United States.

The most frequent complaint offered by these patients was general indisposition, tiredness, exhaustion, lack of energy, in other words — *asthenia.* Fifty-one of the seventy-four gave this as their major complaint. The asthenia is extremely varied in degree and type.

CASE 1—Male; 45; previous history unsuggestive. He complained of "all-tired-out" feeling, a "lead cap" headache and indigestion with distress after eating and loss of appetite.

Physical examination revealed no positive evidence of trouble other than severe pain on deep gentle pressure in the gall-bladder region and a generalized mild icterus. Microscopic examination of feces revealed councilmania.

The patient was given the Weir-Mitchell rest treatment

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